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Longitudinal Changes in Pulmonary Function of Asbestos Workers

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Abstract: Longitudinal Changes in Pulmonary Function of Asbestos Workers: Xiaorong WANG, *et al*. School of Public Health and Primary Care, Chinese University of Hong Kong, Prince of Wales Hospital,China—Objective: To observe the longitudinal changes in pulmonary function associated with exposure to asbestos in a group of highly exposed workers. **Methods:** Pulmonary diffusing capacity (DLco) and spirometry of 243 Chinese asbestos workers were measured and remeasured after 5 and 10 yr. Their annual changes in relation to cumulative asbestos exposure and asbestosis were determined using multivariate analysis. **Results:** The greatest annual decline was observed in DLco, particularly in workers with asbestosis, followed by FVC and FEV_1. A greater decline in DLco accorded with a higher level of cumulative asbestos exposure. A similar trend was also seen in FVC, in which the highest exposure level was associated with 223 m*l* loss over each 5 yr period or 45 m*l*/yr. **Conclusions:** There were substantial declines in DLco and FVC over time in the asbestos workers, and a dose-response trend between asbestos exposure and accelerating functional loss.
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Key words: Asbestosis, Asbestos workers, Longitudinal changes, Pulmonary diffusing capacity, Pulmonary function

China is one of the biggest asbestos producers and consumers in the world. Although mining and use of crocidolite have been banned since 2002, an enormous quantity of chrysotile asbestos continues to be mined and used, with total asbestos production exceeding 400,000 tons in 2008[1)]. It was estimated that there were over 100,000 workers in asbestos mines and asbestos product factories in the country[2)]. The number of exposed workers would be much bigger if those who worked in small asbestos mines and factories as well as those involved in initial sorting and weaving asbestos fibers at home were included[2)]. Given the uncontroversial fact that exposure to asbestos can cause both malignant and non-malignant diseases and the large exposed population, occupational exposure to asbestos will continue to pose a big challenge to occupational and environmental health in China.

Interstitial lung fibrosis, i.e. asbestosis, is a common condition that leads to abnormality of pulmonary function, which is typically characterized as restrictive ventilatory impairment[3,4)]. Existing data also suggest that asbestos workers without asbestosis might have substantially abnormal lung function[5,6)]. The fact that the vast majority of asbestos workers were current or ex-smokers might also lead to airflow obstruction (small airways disease) among them[6,7)]. Therefore, different patterns/profiles of lung function impairment may be presented by asbestos workers in terms of their smoking habits as well as intensity of asbestos exposure.

In this study, we analyzed repeated measurements of pulmonary function tests over 5 and 10 yr among a group of Chinese workers who were heavily exposed to asbestos. The primary objectives were to observe the longitudinal changes in pulmonary function, including both ventilatory function and diffusing capacity, and determine their associations with the intensity of asbestos exposure and radiographically shown pulmonary fibrosis.

Subjects and Methods

The study subjects were selected from an asbestos product manufacturing factory in China, where chrysotile was used to produce asbestos textiles and asbestos building materials, such as asbestos cement and tiles, since the 1950s. The detailed information of the factory and dust concentration measurements have been described elsewhere[6)]. In brief, the total dust concentrations in the work areas always exceeded 3 mg/m^3 and personal sample

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for fibers exceeded 3 fibers/m*l* during the years of factory operation. A baseline survey including questionnaires and pulmonary function testing was conducted on 243 male asbestos workers in 1995. These selected workers represented about 85% of eligible workers who met the inclusion criteria, i.e. either active or retired workers, without overt clinical cardiovascular disorders, neuromuscular dysfunction, or infectious or malignant respiratory disease, other than asbestosis, at the entry time. Their pulmonary function was retested 5 yr (Year_5) and 10 yr (Year_10) later. One hundred twenty five workers returned at Year_5 and 124 at Year_10. Altogether, 174 workers (72%) provided at least two measurements of lung function over the 10 yr.

Trained interviewers using a structured questionnaire collected information on personal characteristics, occupational history, medical history, and smoking habit, along with actual height measurements. Factory records were reviewed to verify the information on job and medical history, when a worker could not recall clearly. Personal cumulative dust exposure up to 1995 was estimated by a calculation based on total dust concentrations at various work areas, which were available at intervals from two to five years, and the sum of each time period of working in a specific work area.

All workers were x-rayed regularly. Asbestosis cases were diagnosed by the Municipal Panel of Pneumoconiosis Diagnosis using the 1986 Roentgeno- Diagnostic Criteria of Pneumoconiosis of China. There were 66 workers diagnosed as having asbestosis at the beginning of the study. These cases were classified as I to II, corresponding to 1/1 to 3/t by the ILO Classification. No new cases were diagnosed during the follow-up period.

Spirometric maneuvers were conducted with a 9-liter water-sealed spirometer (Godart Pulmotest, NV, The Netherlands), following standard methodology[8]. Subjects provided at least three acceptable performances in the standing position with a noseclip. Forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) were derived from the best of three curves for maximal FVC and FEV_1. The ratio of FEV_1 and FVC (FEV_1/FVC%) was calculated. In addition, single-breath carbon monoxide diffusing capacity (DLco) was measured in the sitting position at baseline and Year_5, based on the Epidemiology Standardization Project guidelines[8]. Breathhold time was 10 s, and the washout volume was 1 liter for subjects with a FVC of at least 2 liters and 0.5 liter for subjects with a FVC less than 2 liters. Gas analyses for DLco were performed by a Pulmonary Gas Analyzer (GC-1, China). Tests were performed in triplicate, with an interval of at least 4 min between tests, and the best measurement, with maximal inspired volume and sufficient breathhold time was chosen. All parameters of ventilatory function and DLco were converted to conditions of body temperature and ambient pressure saturated with water vapor (BTPS).

Data analysis of the longitudinal changes in lung function focused on two follow-up periods, i.e., over the first 5 yr and over 10 yr, because DLco measurements were available only over the first 5 yr. The changes in lung function over 5 yr and 10 yr were expressed as annual change percentage (ACP, %), which took baseline values into account, as follows:

$$ACP_{year\_5} = [(PFT_{baseline} - PFT_{year\_5}) /5 \text{ (yrs)} \times PFT_{basline}] \times 100\%$$

$$ACP_{year\_10} = [(PFT_{baseline} - PFT_{year\_10}) /10 \text{ (yrs)} \times PFT_{baseline}] \times 100\%$$

where PFT denotes actual measurements of pulmonary function.

Means and standard deviations were calculated for crude lung function values and annual percentage changes. Then analysis of covariance (ANCOVA) was performed to estimate the adjusted annual percentage change. Two different models were constructed. The first model was to estimate the annual changes adjusted for age, height and smoking in all workers, and workers with and without asbestosis. We tried to include both smoking status and smoking amount (pack-yr) in the models, but found the two variables counteracted each other. Thus, we used smoking amount in the final models, with non-smokers assigned a value of 0. The second model was constructed to evaluate the effects of personal cumulative exposure on the subsequent changes in lung function. Cumulative dust exposures were categorized into quartiles in order to examine a possible exposure-response trend. Adjustments for age, height, smoking and asbestosis were made, and finally, generalized estimating equations (GEEs) were applied to fit the linear regression models with the three points of measurement of FVC and FEV_1 over 10 yr. Predictive variables included personal cumulative dust exposure, smoking, work-year, asbestosis along with age and height. The GEE model takes account of the repeated and correlated measurement design in its covariate structure and makes full use of available data[9]. FVC and FEV_1, as continuous variables, were fitted with an identity link function and exchangeable correlation structure in the GEE models. All analyses were carried out with the Statistical Package for the Social Sciences Software version 15.0 for Windows.

Results

Table 1 shows demographic data and lung function measurements at baseline and the two follow-ups. The age of workers, on average, was 44 yr old (ranging from 20 to 70 yr old) at baseline, and 50 and 55 yr old at Year_5 and Year_10, respectively. Workers' exposure duration was 14 yr at baseline, (ranging from 2 to 34 yr), and cumulative personal dust exposure was 1,373 mg · yr. Smokers accounted for 77% at baseline, and had a slightly

Table 1. Characteristics¶ of asbestos workers at baseline and following years

	Baseline (n=243)	Year_5 (n=125)	Year_10 (n=124)
Age, years	44.3 (15.9)	50.5 (12.1)	55.1 (15.2)
Height, cm	163.6 (6.4)	162.2 (6.3)	162.4 (6.4)
Asbestosis (n, %)	66 (27.2)	50 (40.0)	40 (32.3)
Exposure, years	13.63 (8.47)	18.47 (7.53)	21.64 (8.20)
Smoker (n, %)	187 (77.0)	86 (63.8)	92 (74.2)
Pack-yr§	19.44 (15.09)	28.33 (17.54)	31.58 (18.35)
FVC, m*l*	3,497.2 (786.9)	2,933.4 (718.2)	2,593.6 (913.4)
FEV_1, m*l*	2,638.5 (817.4)	2,105.9 (676.0)	2,000.3 (808.7)
FEV_1/FVC %	74.54 (12.29)	71.28 (12.00)	76.67 (13.26)
DL_{CO}, m*l*/min/mmHg	20.14 (3.77)	15.63 (4.40)	—

¶Values are means and standard deviations (in parentheses), unless stated otherwise. §Calculated among smokers.

Table 2. Baseline data and annual percentage changes (%) of pulmonary function of asbestos workers over follow-up periods

	Baseline	Over 5 yr		Over 10 yr	
		Crude	Adjusted§	Crude	Adjusted
All workers	n=243	n=125		n=124	
FVC, m*l*	3,497.2 (786.9)	–2.98 (2.71)	–2.96 (0.25)	–2.80 (1.74)	–2.68 (0.15)
FEV_1, m*l*	2,638.5 (817.4)	–2.88 (3.70)	–2.65 (0.33)	–2.31 (2.06)	–2.17 (0.20)
FEV_1/FVC %	74.54 (12.29)	0.23 (3.84)	0.49 (0.34)	0.81 (2.27)	0.83 (0.22)
DL_{CO}, m*l*/min/mmHg	20.14 (3.77)	–4.87 (4.64)	–4.94 (0.43)	—	—
Without Asbestosis	n=177	n=75		n=84	
FVC, m*l*	3,726.3 (725.7)	–2.88 (2.57)	–3.01 (0.35)	–2.58 (1.82)	–2.98 (0.18)
FEV_1, m*l*	2,881.6 (768.8)	–3.62 (2.89)	–3.78 (0.44)*	–2.28 (2.09)	–2.54 (0.24)
FEV_1/FVC %	76.81 (12.32)	–0.69 (3.40)	–0.69 (0.46)*	0.51 (2.10)	0.71 (0.26)
DL_{CO}, m*l*/min/mmHg	20.67 (3.75)	–3.94 (5.20)	–4.42 (0.62)	—	—
With Asbestosis	n=66	n=50		n=40	
FVC, m*l*	2,882.9 (594.0)	–3.13 (2.93)	–2.91 (0.43)	–3.27 (1.46)	–3.07 (0.28)
FEV_1, m*l*	1,986.5 (542.6)	–1.78 (4.46)	–1.51 (0.55)	–2.37 (2.04)	–1.80 (0.38)
FEV_1/FVC %	68.44 (9.99)	1.62 (4.07)	1.67 (0.57)	1.44 (2.50)	0.96 (0.41)
DL_{CO}, m*l*/min/mmHg	18.70 (3.45)	–6.14 (3.39)	–5.46 (0.74)	—	—

¶Values are means and standard deviations (in parentheses). §Values are means and standard errors (in parentheses), adjusted by age, height and pack-yr in ANCOVA. *p< 0.01 in comparison with workers with asbestosis.

lower proportion at both follow-up times. There were more smokers among workers without asbestosis than among those with asbestosis at baseline (79% vs. 73%) and at follow-up years. All lung function parameters tended to decrease with follow-up time, except for FEV_1/FVC%, which was elevated at Year_10.

Table 2 shows baseline data and annual percentage change of lung function over 5 and 10 yr. On the whole, the greatest annual decline (5%) was observed in DLco over 5 yr, followed by FVC and FEV_1 at rates of 3%. Not much change was seen in FEV_1/FVC%. The results persisted after adjustment for age, height and smoking. FVC appeared to decline faster than FEV_1 over 10-years. In comparison with workers without asbestosis, those with asbestosis had a faster decline in DLco and slower decline in FEV_1 and therefore FEV_1/FVC% over 5 yr. No significant differences between the two subgroups of FEV_1 and FEV_1/FVC% were found over 10 yr. Both subgroups had about 3% of decline in adjusted FVC over 5 yr and 10 yr.

Table 3 gives the annual percentage changes of functional parameters by cumulative asbestos exposure categorized into quartiles. There was an exposure-response trend in DLco over 5 yr. Greater declines in DLco were in line with higher dust exposures, with rates 47% faster in the second highest exposure and 72% faster

Table 3. Changes in pulmonary function with cumulative asbestos dust exposure over follow-up periods

Cumulative dust exposure	Baseline¶ (n=243)	Adjusted ACP (%)§ Over 5 yr (n=125)	Over 10 yr (n=124)
1st Quartile			
FVC	3,942.4 (687.6)	–2.80 (0.59)	–2.54 (0.40)
FEV_1	3,230.0 (686.4)	–2.59 (0.77)	–2.47 (0.53)
FEV_1/FVC%	82.00 (11.52)	0.52 (0.80)	0.16 (0.57)
DL_{CO}	21.24 (3.63)	–3.50 (1.01)	
2nd Quartile			
FVC	3,814.0 (683.3)	–3.82 (0.50)	–2.47 (0.29)
FEV_1	2,962.0 (692.2)	–3.60 (0.65)	–1.88 (0.39)
FEV_1/FVC%	77.17 (10.06)	0.34 (0.68)	0.73 (0.42)
DL_{CO}	20.54 (3.40)	–4.83 (0.89)	
3rd Quartile			
FVC	3,362.0 (718.1)	–3.24 (0.53)	–3.09 (0.33)
FEV_1	2,388.4 (715.8)	–2.88 (0.70)	–2.64 (0.43)
FEV_1/FVC%	70.40 (11.45)	0.58 (0.73)	0.92 (0.47)
DL_{CO}	19.90 (3.68)	–5.16 (0.94)	
4th Quartile			
FVC	2,859.9 (564.7)	–2.09 (0.53)	–3.05 (0.35)
FEV_1	1,962.6 (503.8)	–2.41 (0.70)	–2.21 (0.47)
FEV_1/FVC%	68.49 (11.36)	–0.34 (0.73)	1.36 (0.51)
DL_{CO}	18.83 (4.02)	–6.01 (0.90)	

¶Values are means and standard deviations (in parentheses). §Values are means and standard errors (in parentheses), adjusted by age, height, pack-yr and asbestosis in ANCOVA.

Table 4. GEE analysis¶ of longitudinal changes in FVC and FEV_1 over 10 yr

	FVC, m*l*	FEV_1, m*l*
Cumulative dust exposure		
1st Quartile	0	0
2nd Quartile	83.28 (–89.39, 255.94)	97.94 (–83.19, 279.07)
3rd Quartile	– 23.04 (–245.46, 199.38)	15.36 (–193.50, 224.22)
4th Quartile	– 222.83 (–501.89, –56.22)	– 95.23 (–339.86, 149.41)
Age	– 17.12 (–24.27, –9.97)	– 30.98 (–37.34, –24.63)
Height	53.54 (42.45, 64.62)	38.50 (28.68, 48.31)
Pack-year	– 0.17 (–5.66, 5.31)	– 2.27 (–6.71, 2.17)
Asbestosis	– 161.34 (–346.54, 23.86)	–5.39 (–162.12, 151.34)

¶Using actual values of the three measurements in GEE models; values are estimates and 95% confidence interval (in parentheses).

in the highest exposure, relative to the lowest quartile of cumulative dust exposure. Although the trend was less consistent in FVC over 5 yr, FVC tended to decline faster with increased exposure levels over 10 yr. Trend tests showed none of the comparisons was statistically significant. Age and height were significant contributors to the changes in all of the functional parameters. Pack-year and asbestosis were positively related to functional loss, though not statistically significant.

Longitudinal changes in FVC and FEV_1 were evaluated with GEE analysis that took into account all three values over 10 yr. As shown in Table 4, a trend indicating decline of FVC with increasing cumulative exposure was found. The highest exposure level contributed to FVC decline by 223 m*l* each 5-year period or 45 m*l*/yr. On the other hand, a less consistent trend was seen for FEV_1, but the highest exposure level was associated with the greatest decline. Asbestosis, increased smoking pack-years and

age were factors that contributed to greater declines in both FVC and FEV_1, whereas height was inversely related to their declines. Work-years was a significant contributor to the decline in FVC (–12.9 m*l*; 95% CI: –21.47, –4.36) and in FEV_1 (–8.62 m*l*, 95% CI: –14.62, –2.63) when the variable of cumulative dust exposure was excluded from the models. A similar analysis was performed for DLco, for which only two values were available. Its association with cumulative dust exposure was similar to that observed for FVC. In addition, each work-year was associated with declined DLco by 0.07 m*l*/min/mmHg (95% CI: –0.12, –0.01) when cumulative dust exposure was removed.

Discussion

Occupational exposure to asbestos has been and will continue to be a major occupational health problem in China. Overall exposure levels in asbestos mines and factories were surprisingly high prior to 2,000[10], although industrial hygiene in workplaces has much improved in recent years. In the present study, we observed workers who were heavily exposed to chrysotile asbestos, with 14 yr of average exposure at the baseline. The concentrations of total asbestos dust at the workplace were always higher than the national standard (2 mg/m^3) applied prior to 2002.

Several interesting findings were derived from the data analysis. First, we observed that the most rapidly declining parameter was DLco in these workers. As a whole group, an annual decline in DLco of 5% over 5 yr was observed, even after factors such as age, height and smoking were taken into account. Workers with asbestosis showed greater reductions in both crude and adjusted values of DLco than those without asbestosis. Furthermore, the loss rate of DLco was consistent with increasing cumulative exposure. In comparison with the lowest category of cumulative dust exposure, the second highest exposure level showed a 47% faster decline, and the highest exposure level a 72% faster decline. The absence of significant differences in these comparisons could be due to inadequate statistical power because of a small sample size in the subgroup analyses. DLco has been found to be a sensitive parameter for functional deficit in asbestos-exposed workers and asbestosis patients[5, 11, 12]. Picado and coworkers[13] suggested that DLco changes do not regress, therefore progressive reduction in DLco might be a reliable finding for the diagnosis of asbestosis. One study observed a nine-year change in DLco in a small number of asbestos workers, and found that its decrease correlated well with parenchymal abnormalities[11]. Although epidemiological studies have shown that DLco is a very useful index in evaluating functional changes in asbestos workers, few studies have determined its longitudinal changes, partly because measurement of DLco requires sophisticated equipment and techniques, which may not be always feasible, especially in fieldwork. Despite the fact that only five-year data were available in the current study, the data show the longitudinal changes in DLco were associated with pulmonary interstitial fibrosis, and with intensity of asbestos exposure. This confirms that longitudinal observation of DLco in asbestos workers is of a great value in monitoring asbestos-induced functional deficits.

Accelerated annual losses of FVC and FEV_1 have been reported in several longitudinal studies of asbestos workers exposed to relatively low levels of asbestos[14], or those who had retired from asbestos processing[15]. In our study, the adjusted annual loss was 3% for FVC and 2.7% for FEV_1 over 5 yr, and the declines were slightly slower over 10 yr. Aging is a recognized confounding factor of longitudinal declines in pulmonary function. The observed changes in FVC and FEV_1, as well as DLco might be partly explained by the aging effect, as there was no a parallel control group available consisting of workers without exposure to asbestos. However, the observed longitudinal declines could not be entirely ascribed to the aging effect, since the GEE analysis using three data points revealed that FVC and FEV_1 declined faster with cumulative exposure after adjusting for potential confounding factors, including age. Also, asbestosis was found to be associated with a greater decline in FVC in the GEE model, an excessive 161 m*l* decline over 10 yr or about 16 m*l*/yr, which was larger than in those without asbestosis. On the other hand, no obvious differences in FEV_1 and FEV_1/FVC% were observed between those with and without asbestosis. There were no consistent changes in the two parameters with cumulative exposure, either. Our present results agree with previous findings indicating a restrictive functional impairment associated with asbestos exposure[3, 6, 16]. Yet, the difference in FVC was not significantly different between the workers with and without asbestosis, a result which might be due to inadequate statistical power. Another possible explanation is that the majority of the workers with asbestosis stopped working in the dusty environment once the diagnosis was made, whereas many of the workers without asbestosis continued working during the observation period. The association between accelerated decline in FVC and FEV_1 and current asbestos exposure was documented in a previous study[15]. All of these factors might have contributed to reducing the differences between workers with and without asbestosis.

Previous studies have shown that smoking is a major risk factor of obstructive airway limitation in asbestos workers, which is mainly expressed in decreased FEV_1 and FEV_1/FVC%[3, 6]. Longitudinally, it has been shown that smoking accelerates the rate of decline in the general population[17] and in asbestos workers[16]. In this study, either smoking or smoking amount was related to a faster

decline in lung function over time. A lack of statistical significance for smoking effect might be attributable to the majority of the workers in this study being smokers, which might have obscured the smoking effects. In addition, the proportion of smokers among the workers without asbestosis was higher in those with asbestosis at baseline and the follow-up years. This might explain why we observed a greater decline in FEV_1 in the workers without asbestosis.

Several limitations of this study should be noted. There was a proportion of the workers who were lost to follow-up at both 5 yr and 10 yr, which might be a source of selection bias if those lost to follow-up were differential. In order to explore the extent to which the results might be biased by those lost to follow-up, we carried out a sensitivity analysis by comparing lung function tests between those who returned and those who did not. At the 5-year follow-up, there were differences in baseline age, cumulative exposures and smoking amount between those followed and those lost to follow-up. Accordingly, the baseline values of lung function parameters were lower in those followed than those lost to follow-up, which might have led to a somewhat overestimated decline in lung function over the first 5 yr. At the 10 yr follow-up, there were no significant differences between the two subgroups in either baseline data or the 5 yr data. This implies that the results over the second 5 yr of follow-up were not substantially affected by those lost to follow-up. Another limitation of this study was the small sample size, particularly in the subgroup analyses, which provides a possible explanation for the non-significant differences found in the comparisons of repeated measurements made in different years, and between workers with and without asbestosis.

Conclusions

Abnormal pulmonary function is the most common adverse health outcome resulting from asbestos exposure. Yet, longitudinal data of ventilatory function in asbestos workers is sparse, not to mention data of pulmonary diffusing capacity. This study has provided additional information illustrating the longitudinal changes in both lung diffusing capacity and ventilatory function of workers who were highly exposed to asbestos. The results suggest a dose-response trend between the intensity of asbestos exposure and accelerating functional loss of asbestos workers.

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